Hyperbaric oxygenation treatment of acute paraplegia after resection of a thoracoabdominal aortic aneurysm

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Acute spinal cord ischemic injury after resection of thoracoabdominal aneurysm remains a relatively common and potentially devastating complication. The complete resolution of postoperative paraplegia after resection of a type II thoracoabdominal aneurysm, after treatment with hyperbaric oxygenation, is reported. (J Vasc Surg 1999;30:1158-61.)

Acute paraplegia remains a potentially debilitating complication of thoracoabdominal aortic resection, despite a number of intraoperative techniques designed to prevent ischemic spinal cord injury. Once injury has occurred, treatment options are limited. Recovery from paraplegia is often prolonged, dependent on natural resolution of reversible processes, and frequently incomplete, with a variable degree of residual deficit in most cases. In this case, acute paraplegia was treated by means of hyperbaric oxygenation (HBO), with rapid and complete resolution.

CASE REPORT

During an investigation for refractory hypertension in a 63-year-old man, who had a tube graft replacement for an infrarenal abdominal aortic aneurysm 7 years earlier, a nonfunctioning, atrophic left kidney associated with renal artery occlusion and a suprarenal aortic aneurysm, 4.8 cm in diameter and involving the coeliac axis, superior mesenteric, and renal arteries, were revealed. After 2½ years of computerized tomographic (CT) surveillance, the aneurysm increased dramatically, to a diameter of 7.8 cm in a 6-month interval. A type II thoracoabdominal aneurysm, extending from just beyond the left subclavian artery origin to the old infrarenal aortic graft, was shown by means of additional CT scanning.

With the patient under general anesthesia, the aneurysm was exposed retroperitoneally via an oblique thoracoabdominal incision through the left eighth intercostal space, with division of the diaphragm and supplementary thoracotomy through the fifth intercostal space for proximal exposure. The aneurysm was replaced by using a gelatin-coated woven Dacron tube graft, 25 mm in diameter, from the T4-5 level to the old infrarenal aortic graft, with a sequential clamp-and-sew technique and systemic heparinization (340 IU/kg body weight). During segmental proximal clamping, the distal aorta and its visceral and intercostal branches were perfused with oxygenated femorofemoral bypass via the left common femoral artery and vein. A variable flow rate of 1.5 L to 2.5 L per minute, at a mean pressure of 40 to 50 mm Hg, was maintained by using a pediatric-sized membrane oxygenator with a roller pump. Hypotension in the arch vessel distribution during distal femorofemoral perfusion was prevented by increasing central venous perfusion with autotransfusion, adjunctive cryoprecipitate, and platelet and crystalloid infusion, and by manipulating venous return to the pump oxygenator. No other adjunctive spinal-cord protective modalities were used.

Two widely patent intercostal arteries at the T10 level with good back-bleeding were anastomosed in situ to an oval window in the graft, with an unperfused clamp time of 10 minutes. There were no other patent intercostal arteries arising from the aneurysmal sac. The coeliac axis, superior mesenteric, and right renal arteries were anastomosed in situ to a boomerang-shaped window in the graft, with an unperfused clamp time of 20 minutes. The left kidney was not revascularized because of its lack of function, atrophy, and long-standing renal artery occlusion. Blood loss for the procedure was 3.5 L, which was saved, washed, and retransfused with a cell-saver. The patient's early postoperative recovery was satisfactory, with stable blood pressure, minimal blood drainage, adequate urine output with a serum creatinine level of 1.1 mmol/L, and no neurological sequelae in the lower limbs. Results of postoperative continuous monitoring of arterial blood pressure, heart rate, electrocardiography, cardiac output, pO_2, pCO_2, urine output, intravesical pressure, and core temperature remained stable, with no abnormalities detected.

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Eighteen hours after operation, complete paralysis, which had appeared suddenly in the interval between successive 30-minute neurological observations, developed in both legs of the patient. All sensation was lost below the T9 level. Dexamethasone (20 mg) was given, and a regimen of intravenous methylprednisolone was instituted (30 mg/kg loading dose, followed by 5.4 mg/kg by infusion for 48 hours). In the absence of any abnormalities detected by means of monitoring, a diagnosis of postischemic cord edema with compression ischemia of the lateral columns was considered most likely, and we decided to treat it with HBO. To prevent eardrum perforation, a Grommet vent was inserted in each ear. HBO was started 4 hours after onset of paralysis, with the patient breathing 100% oxygen for 90 minutes at a pressure of 242 kPa (2.4 atmospheres absolute). After treatment, there was a dramatic improvement in both motor and sensory deficits, with residual weakness in the proximal muscle groups and paresthesia in both legs. Two additional similar treatments during the next 24 hours resulted in complete clinical resolution of the paraplegia. Full mobilization on the fourth postoperative day was, however, associated with an unsteady gait and proximal muscle weakness when walking short distances. Seven days after operation, the patient experienced impaired bladder emptying, and "burning" dysesthesia developed in both feet. He underwent three additional treatments with hyperbaric oxygenation, which resolved the bladder problem and left him with minor paresthesia in both feet and ankles in a bobby-sock distribution. There were no other complications from the surgery. He was discharged from the hospital on the 18th postoperative day. At a 4-week follow-up examination, the patient's motor function was normal, with minor residual paresthesia on the soles and dorsum of his feet. At an 8-week follow-up examination, complete clinical recovery was documented by an independent specialist neurologist.

DISCUSSION

Acute paraplegia or other symptoms of spinal cord ischemia after thoracoabdominal aortic aneurysm replacement is not uncommon, having a reported incidence of 5% to 40%.[1,2] A variety of methods for intraoperative spinal cord protection, including cerebrospinal fluid (CSF) drainage, cord hypothermia, adjunctive pharmacology, reimplantation of intercostal arteries, and distal aortic perfusion, have been advocated. However, the effectiveness of these prophylactic modalities cannot be predicted, and their individual benefits have not been conclusively determined.[3-6] There is no definitive postinjury treatment that guarantees improvement or resolution, except that of the natural progress of the lesion, and complete resolution is uncommon.

The natural history of ischemic spinal cord injury after aortic resection is variable, with differing degrees of motor and sensory deficit. Motor deficit is related to loss of perfusion in the anterior spinal artery distribution. Variable sensory deficit is possibly related to secondary ischemia of the lateral and posterior columns of the cord, because of compressive cord edema and decreased net spinal cord perfusion resulting from the loss of arterial perfusion pressure and increased CSF pressure. (Spinal cord perfusion pressure is the result of a balance between arterial perfusion pressure and CSF pressure.)

Immediate postoperative paraplegia is directly related to decreased perfusion and increased CSF pressure, which results in the death of some or all cells in the ischemic area. Such injury is not capable of complete resolution, but, in some cases, the recovery of surviving cells is possible in a protracted period, with residual deficit. Delayed onset paraplegia, on the other hand, is probably related to the effects of ischemia-reperfusion, with resultant free radical formation, cord edema, and possibly associated increased CSF pressure. The prospect of reversal of such effects and preservation of neuronal cells with recovery exists, provided effective therapy is implemented before secondary compressive irreversible ischemic neuronal damage supervenes. Hill et al[7] have reported reversal of delayed-onset paraplegia with CSF drainage after repair of type I thoracoabdominal aneurysms in two patients. They attributed the clinical improvement to a possible improvement in spinal cord circulation associated with decreased CSF pressure.

Hyperbaric oxygen therapy has been studied in animal models of spinal cord injury. Type III cord injuries induced in rats have shown improvement after HBO therapy, which appears to arrest the spread of hemorrhage and resolve edema.[8] A study of patients with primary nonischemic spinal cord injury showed that those patients treated with HBO appeared to recover more quickly, although their final motor scores were similar to those of patients who received nonspecific conventional treatment,[9] suggesting that HBO facilitates recovery of reversibly damaged cells but has no benefit for irreversibly damaged cells. HBO has been advocated in a number of clinical situations associated with ischemia-reperfusion injury. Although its role in free radical formation and antioxidant activity is not completely understood, HBO is believed to enhance antioxidant defense mechanisms. It is beneficial in ischemia-reperfusion injury through the mechanism of inducing arterial vasoconstriction, which reduces tissue edema while maintaining tissue oxygenation.[10] Decreased perfusion associated with vasoconstriction is more than compensated for by the increased

solubility and carriage of oxygen and a greatly increased tissue diffusion gradient for oxygen in hyperbaric conditions. Increased tissue oxygenation stabilizes the neuronal cellular membrane sodium pumps and calcium channels, preventing sodium and calcium accumulation in the cell and potassium loss from the cell, which in turn prevents cell death. With postischemic spinal cord edema, the effects of HBO in reducing cord edema and maintaining tissue oxygenation is also associated with decreased CSF pressure. These combined effects maintain neuronal cellular integrity, prevent secondary vascular compressive ischemia, and improve net spinal cord perfusion pressure. The value of HBO in the resolution of paraplegia is thus critically related to its implementation before irreversible ischemic damage occurs. Its value in acute paraplegia caused by direct intraoperative ischemic injury is likely to be dependent on the number of irreversibly damaged cells balanced against the number of reversibly affected cells. It could be speculated, however, that HBO may not be associated with complete resolution in early-onset primary paraplegia, in contrast with delayed-onset secondary paraplegia.

In the case reported, the delayed onset at 18 hours after operation was unexpected, particularly when the intercostal arteries were widely patent, with good back-bleeding and the unperfused clamp time of only 10 minutes. The back-bleeding during anastomosis was not controlled, and, in retrospect, the venting effect of the back-bleeding may have resulted in loss of perfusion of the spinal cord. Our future practice will involve occluding the intercostals during anastomosis, so that perfusion pressure through the collateral circulation is maintained. The dramatic improvement of paraplegia after the first treatment with hyperbaric oxygen also raises the possibility that HBO administered immediately after operation may represent a prophylactic adjunctive therapy in patients with preinfarction or pre-edema spinal ischemia, and we are considering implementing this as a routine in future practice. Although we are unable to define whether hyperbaric oxygenation improved this patient more than spontaneous resolution, we feel that, in the face of the severity of the paraplegia, improvement after 90 minutes of therapy and final complete recovery are well in advance of the expected norms for spontaneous resolution.

It is possible that the patient may indeed have had acute-onset primary paraplegia 18 hours after operation caused by thrombosis of the re-anastomosed intercostal arteries, rather than delayed-onset paraplegia caused by ischemia-reperfusion injury. However, the process causing paraplegia was clearly reversible in view of the clinical recovery, and such an outcome would have been unlikely if the primary process had been occlusion of the only patent intercostal vessels. Although the definitive diagnosis of spinal cord edema with magnetic resonance imaging (MRI) would have confirmed the clinical diagnosis, there was nothing to be gained for the patient, and the delay incurred in gaining evidence of cord edema by means of MRI may have resulted in some permanent damage. In retrospect, we would opt for early HBO therapy, rather than confirmation by means of MRI imaging, should a similar event arise in the future, because clinical diagnosis in the time-critical situation should be acted on in the patient's best interests, rather than in the interest of exhaustive documentation.

Finally, HBO is not available in all institutions. We are fortunate to have a long-established (30-year) HBO clinical service, with a large chamber capable of receiving an in-bed patient with full monitoring and medical and nursing attendants. The facility is situated adjacent to the emergency department, the intensive care unit, and the operating suites. The use of this therapy is a routine part of our vascular service and does not represent an extraordinary nor logistically difficult therapy. Clearly, in institutions without a hyperbaric oxygen facility, delays in transferring a patient to another facility would almost certainly preclude HBO as an effective therapy. In this situation, rapid on-site implementation of other therapies, such as CSF drainage and pharmacological adjunctive therapy, should remain the primary emergency option. However, transfer to an HBO facility after such therapies have been instituted may possibly still have a role in minimizing the final deficit and shortening the recovery period. The result in this case suggests that HBO, when readily available, should be considered in all patients with postoperative spinal cord injury and implemented as soon as possible after onset of paraplegia.

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